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Early extrastriate activity without primary visual cortex in humans

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Abstract

Damage to the primary visual cortex (V1) destroys the major source of anatomical input to extrastriate cortical areas (V2, V3, V4 and V5) and produces cortical blindness – an absence of any sensation of light and colour – in the visual field contralateral to the side of the lesion. Neuroimaging studies, nevertheless, have recently demonstrated dorsal and ventral extrastriate activation for stationary stimuli presented to the blind visual field in the absence of V1 activity in human subjects. To clarify the moment in time that visual information reaches extrastriate areas, by means of event-related potentials (ERPs) we tracked the temporal course of responses to complex visual stimuli (faces) presented in the blind field of a hemianopic patient. Stimulation of the normal visual field elicited a positive occipital deflection (P1) at 140 ms. A P1 response was also observed with stimulation of the blind field, although slightly delayed (20 ms) and reduced. Its topography and timing demonstrate that early neural activity for stationary stimuli takes place within extrastriate regions despite V1 denervation.

Keywords: Primary visual cortex; Event-related potentials; Positive occipital deflection; Extrastriate cortex; Blindsight

Damage to the primary visual cortex produces hemianopia, a homonymous visual field deficit contralateral to the site of the lesion. Nevertheless, numerous studies have described a wide range of unaware visual functions in the blind field of such patients in the absence of awareness, such as detection and spatial localization by eye and hand movements of stationary or moving stimuli [6,12] or discrimination based on line orientation or wavelength [14]. The term 'blindsight' [20] has been given to these unaware residual visual functions, which suggest that indirect subcortical routes from the retina and the lateral geniculate nucleus (LGN) reach several extrastriate areas bypassing the primary visual cortex (V1).

Recent neuroimaging studies have demonstrated that stimulation of the visually blind hemifield of blindsight patients with stationary complex visual stimuli (coloured drawings of natural objects) yields activation of occipito-temporal regions [15], extending earlier report of dorsal extrastriate activity during presentation of moving stimuli [3]. The ventral extrastriate regions included V4 and LO, a human visual cortex area dedicated to shape perception [8]. Importantly, there was no activation in the damaged or deafferented area V1 on the lesioned side of the brain, even when reversing checkerboard patterns were presented.

However, the low temporal resolution of functional magnetic resonance imagery (fMRI) does not allow to ensure that these residual visual activities rapidly follow stimulus presentation or are due to slower 'top-down' processes such as mental imagery [11]. More generally, no information is yet available regarding the moment in time visual information reaches extrastriate areas in the absence of primary visual cortex. To study this issue, we recorded event-related potentials in a hemianopic patient presented with complex objects (faces) alternatively to his normal and blind field.

The subject was patient G.Y., a well-studied 43-year-old hemianopic patient with damage to the left medial occipital lobe resulting in a right half field of blindness (except for a small region of 3.5° of macular sparing), whose residual visual abilities have been studied in many previous behavioural and neuroimaging studies [2,5,7,9,18,19]. He was tested four times in a dimly lit, electrically shielded room with the head restrained by a chin rest at 60 cm of the screen, fixating a cross on the centre of the screen. For the main experiment, he was presented with two types of stimuli:

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achromatic upright faces and inverted faces. All types of stimuli appeared randomly either in the good visual field or in the affected field (80 in each field, 2 averages) and were completely randomized. Size of the stimulus was 5.5 cm width (5.4°) and 8.5 height. Total luminance of the stimulus was 25 cd/m^2 (screen background and room luminance: 1 cd/m^2). Faces were presented with the nearest edge of the stimulus at least 4.7° removed from the central fixation cross. G.Y.'s task was simply to press one response key when he saw a normal face and another key when he perceived an inverted face. For all stimuli presented in his blind hemifield, he was encouraged to guess. Stimulus duration was 250 ms and intertrial interval was randomized between 500 and 650 ms. The same experiment was repeated 2 months later with stimuli presented for 1250 ms (averages 3 and 4). In another experiment, four types of stimuli (250 ms) were presented: achromatic upright faces and inverted faces and red coloured upright and inverted faces (average 5–8). G.Y. was also tested in a task in which he had to discriminate photographs of cars from faces (250 ms presentation; averages 9 and 10). Finally, G.Y. was presented with 5 blocks of emotional faces (1250 ms presentation time) and he had to guess the emotional content of the faces (happy vs. fear) by pressing one of two keys (averages 10–15). Over the complete set of experiments, 15 different averages were computed for faces presented in his normal and blind field.

Although G.Y. is highly experienced and reliable at maintaining fixation in these kinds of tasks [3,11], electrooculogram (EOG) was recorded bipolarly from electrodes placed on the outer canthi of the eyes, and in the inferior and superior areas of the orbit. Scalp electroencephalograph (EEG) was recorded from 58 electrodes mounted in an electrode cap. Recordings were performed with a left mastoïd reference in the main experiment. EEG was amplified with a gain of 30 K and bandpass filtered at 0.01–100 Hz. Electrode impedance was kept below 5 kΩ. EEG and EOG were continuously acquired at a rate of 500 Hz (Neuroscan). After removal of EEG and EOG artefacts, epochs beginning 100 ms prior to stimulus onset and continuing for 924 ms were made. They were referenced off-line to a common average reference. All the data were also referenced to a centro-frontal single electrode (FZ). The data were low pass filtered at 30 Hz. Peak amplitude and latencies of the P1 at selected electrodes were measured relative to a 100 ms pre-stimulus baseline. Source estimation (Advanced Source Analysis; ©1999, A.N.T. Software BV; see Ref. [13]) was performed on the early dipolar complex (time window 20 ms before the P1 peak) with a single unconstrained dipole.

In the first experiment, G.Y. accurately discriminated the orientation of the stimuli presented in his intact visual field (99%, 1001 ms) but he was at chance in his blind field. Visual stimulations in the left, normal field gave rise to a dipolar complex consisting of a left occipital positivity (P1) associated with a negative counterpart at right centro-lateral electrodes (Figs. 1 and 2). Source localization confirmed the right extrastriate origin of this complex [10]. For stimulations in the normal field, the mean coordinate of the source in the realistic head shape model (© 1999, A.N.T. Software BV) was: $-72x$ $-20y$ $17z$ (x is antero-posterior, y is left-right and z is vertical; 9.19% residual variance).

Stimulation in the blind field led to a similar dipolar complex consisting of an occipital positivity associated with a negative counterpart at left centro-lateral electrodes (Figs. 1 and 2). It was delayed and reduced (20 ms later; Fig. 1) compared with the activity observed for stimulation in the good field. Its topography shows a slightly less lateralized distribution at posterior sites (Fig. 2). Source localization identified a single dipole in left extrastriate cortex ($-61x$ $21y$ $20z$; 11.8% residual). Similar topographies (see Fig. 2; right part) and identical results for source localization were obtained with the data referenced to the FZ electrode.

Although G.Y. was at chance for all the tasks he had to perform on the faces, all 15 averages of waveforms obtained with the various stimulations in the blind field led to the presence of an occipital P1 component which was delayed (grand averages: 140 ms for normal field, 160 ms for blind field; $t_{14} = 6.46$, $P < 0.001$) and reduced (grand averages: 6.05 μV for normal field, 4.03 μV for blind field; $t_{14} = 5.057$, $P < 0.001$) as compared with the earliest activity evoked by normal field presentations. Non-face stimuli (cars) also generated a substantial P1 when presented to G.Y.'s blind field.

This is the first report of a rapid extrastriate cortex activation despite V1 denervation for stationary stimuli. Without the earliest contribution from the primary visual cortex, the P1 is reduced and delayed but still substantial (Fig. 2). These data are consistent with the extrastriate activation evidenced in fMRI studies [11,17] for blind visual field stimulations but adds temporal information unavailable using fMRI. The topography and latency of P1 observed here show that these

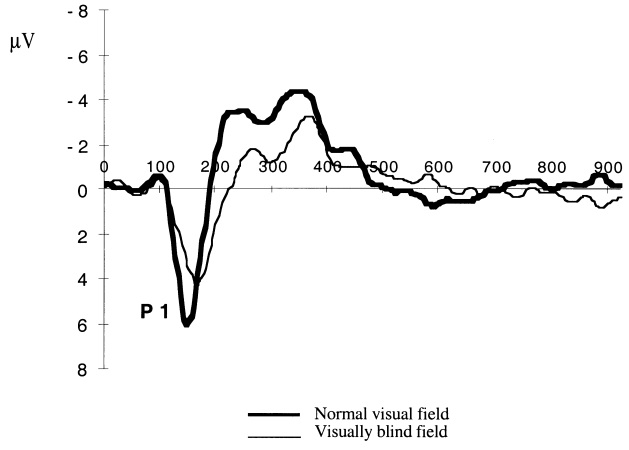

Fig. 1. Evidence for a reduced an later visual P1 activity when G.Y. is stimulated in his blind visual field. The grand average waveforms (15 experiments, electrode OZ, system 10–20) shown are obtained for face stimuli presented in the normal and blind visual field of patient G.Y. with complete left hemisphere V1 denervation.

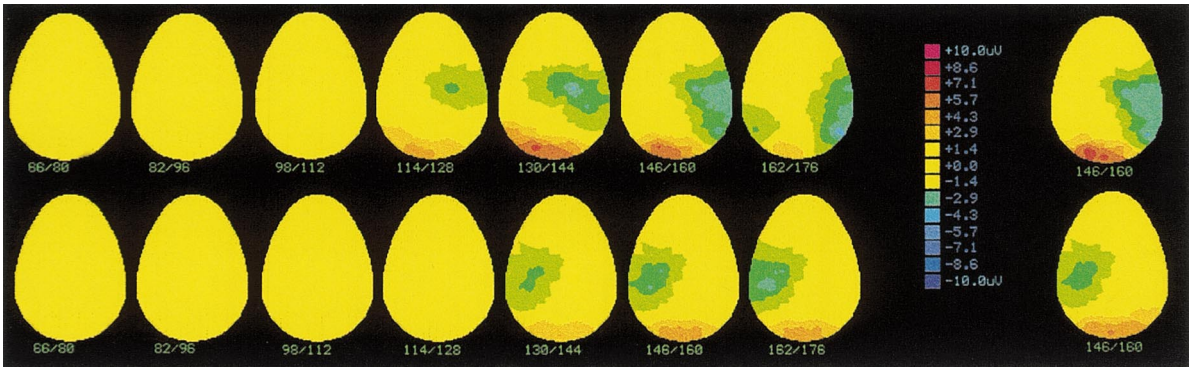

Fig. 2. Above: topography of the grand average (15 experiments, cut-off: −2 μV/2 μV) from 66 to 176 ms, showing the P1/N1 component to stimuli presented in the good (left) visual field of G.Y. Below: the evidence of the early extrastriate activity (P1/N1) observed when G.Y. is stimulated in his blind visual field. The occipital positivity observed for stimulations in the normal visual field is earlier and larger at ipsilateral sites (above) than at contralateral sites. By contrast, stimulations in the blind hemifield led to a bilateral positivity, that is reduced and delayed as compared with the neural activity observed for normal visual field stimulations. The topography on the right is obtained with the centro-frontal electrode (FZ) as reference electrode.

activations do not come from top-down processes such as visual imagery of complex objects activating visual extrastriate areas [11] but rather reflect bottom-up flow of information to extrastriate regions bypassing V1.

In the different ERPs experiments conducted with G.Y., we modified the following parameters: face orientation, presentation time, object-type: faces vs. cars, and colour of the stimulus. Some of these parameters (face orientation, stimulus type) affected the potentials following the P1 when the normal field was stimulated. However, there was no obvious evidence of such variability with blind field stimulations, nor was there any evidence that G.Y. could carry out the complex visual discriminations (upright vs. inverted; face vs. cars; fearful vs. angry faces) better than at chance levels. The absence of any difference between red-coloured and black-and-white stimuli when presented to the blind field suggest further that the pathways that might carry colour information from the LGN to extrastriate areas such as V2 and V4 (see Ref. [16]), may not be the main routes involved in the observations reported here. However, these observations are in line with the fact that other direct subcortical projections reach several extrastriate areas bypassing V1, including indirect subcortical routes from the retina to the pulvinar (directly and also via the superior colliculus) and the LGN, or other subcortical projections [2,20]. The areas contributing to an early positive potential with an occipital topography may include V5 and dorsal V3, in which activations have been previously observed in G.Y.'s brain by positron emission tomography [3] and which can be rapidly activated by motion stimuli despite V1 lesions [1] in the monkey, but also V2d, V3a and V4v in which residual activity has been recently evidenced for stationary stimuli using fMRI [4,11].

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